

ORIGINAL ARTICLE

# Pregabalin poisoning and rising recreational use: a retrospective observational series

Katherine Z. Isoardi<sup>1,2,3</sup>  | Gregory Polkinghorne<sup>1,2</sup> | Keith Harris<sup>1,2</sup> |  
Geoffrey K. Isbister<sup>3,4</sup> 

<sup>1</sup>Clinical Toxicology Unit, Princess Alexandra Hospital, Brisbane, Queensland, Australia

<sup>2</sup>Faculty of Medicine, University of Queensland, Brisbane, Queensland, Australia

<sup>3</sup>Clinical Toxicology Research Group, University of Newcastle, Newcastle, New South Wales, Australia

<sup>4</sup>Department of Clinical Toxicology and Pharmacology, Calvary Mater Newcastle, Newcastle, New South Wales, Australia

**Correspondence**

Dr Katherine Isoardi, Clinical Toxicology Unit, Princess Alexandra Hospital, Ipswich Rd, Woolloongabba, QLD 4102, Australia.  
Email: katherineisoardi@yahoo.com.au

**Funding information**

National Health and Medical Research Council, Grant/Award Number: Senior Research Fellowship ID1061041; NHMRC Senior Research Fellowship, Grant/Award Number: ID1061041

**Aims:** With rising use worldwide, pregabalin is increasingly implicated in poisoning deaths. We aimed to investigate the clinical effects and complications of pregabalin poisoning.

**Methods:** This is a retrospective review of patients presenting with pregabalin poisoning to two tertiary toxicology units from 1 July 2014 to 30 June 2019. Patients were identified from prospective databases maintained by both units and data were extracted from these in addition to medical records.

**Results:** There were 488 presentations in 413 patients (237 [57%] male) over the five-year period. The median age was 41 years (IQR 31–50 years). Deliberate self-poisonings accounted for 342 (70%) presentations, with 121 (25%) recreational exposures. Recreational exposures increased over the period from 2 (4%) in the first year to 54 (39%) presentations in the final year. The median dose of pregabalin was 1200 mg (IQR 600–3000 mg, range 75–16 800 mg). Co-ingestions occurred in 427 (88%) presentations, with sedating agents being co-ingested in 387 (79%)—most commonly opioids and benzodiazepines in 201 (41%) and 174 (36%) presentations respectively. Coma (GCS < 9) occurred in 89 (18%) cases, with 52 (11%) patients being intubated. Only one (0.2%) of these patients had not co-ingested a sedating agent. Hypotension occurred in 26 (5%) cases, all with co-ingestants. Seizures occurred in 11 (2%) cases, 3/59 (5%) in pregabalin-only overdoses. The median length of stay was 16.5 hours (IQR 10–25 hours).

**Conclusions:** Pregabalin overdose does not cause severe toxicity, but rather mild sedation and, uncommonly, seizures. Coma is common in the presence of sedating co-ingestants. Recreational use is increasing.

**KEY WORDS**

drug abuse, overdose and poisoning, toxicology

## 1 | INTRODUCTION

Pregabalin is a gamma-aminobutyric acid analogue that binds to voltage-gated calcium channels to inhibit the release of excitatory

neurotransmitters, which accounts for its antinociceptive, anticonvulsant and anxiolytic properties.<sup>1,2</sup> Over the last decade, pregabalin has been increasingly prescribed for neuropathic pain, as well as a number of off-label indications including anxiety, mood instability and withdrawal syndromes.<sup>1–4</sup>

With the broadening of both approved and off-label indications, pregabalin use has increased worldwide over the last

The authors confirm that the PI for this paper is Katherine Isoardi and that she had direct clinical responsibility for patients.

decade.<sup>4–6</sup> As with most medications, this leads to increasing rates of poisoning,<sup>7</sup> both deliberate overdose and recreational use.<sup>8,9</sup> The potential for pregabalin to be used recreationally is being increasingly recognised. Euphoria occurs with therapeutic use,<sup>10</sup> but tolerance develops rapidly. This drives continued and escalating use.<sup>1</sup> People with pre-existing substance use disorders are particularly vulnerable.<sup>2,11</sup>

There is limited literature describing the clinical effects of pregabalin poisoning despite its greater availability and increasing recreational use. One case report of a large overdose and a small case series of 23 pregabalin ingestions report minimal clinical effects or complications.<sup>12,13</sup> Reported symptoms of intoxication include sedation, dissociation, relaxation, numbness, disinhibited behaviour, empathy and hallucinations.<sup>2</sup> However, coma and respiratory depression have been reported,<sup>14</sup> particularly with sedating co-ingestants.<sup>1,15</sup> It is unclear whether pregabalin plays a role in the toxicity with sedative co-ingestants, but it is being increasingly implicated in poisoning deaths.<sup>4,8,9,11</sup>

We aimed to describe the clinical features and complications of pregabalin overdose, including the potential role of co-ingestants and comparing deliberate self-poisoning with recreational use.

## 2 | METHODS

### 2.1 | Study design and settings

This is a retrospective review of adult (>15 years) patients with pregabalin poisoning presenting to two toxicology units. Both units manage all poisoned patients that present to their emergency department from their draining geographical catchment area. One is located within a tertiary hospital with an emergency department that has approximately 64 000 presentations annually. The other is within a metropolitan hospital, with an emergency department that has approximately 38 000 presentations annually. The toxicology units admit approximately 2000 and 900 patients each year respectively. Data on every patient presentation to each unit are prospectively recorded in a purpose-built relational database that undergoes weekly audit and data review. Admission data are collected on either a preformatted admission sheet or tablet-based application.<sup>7,16</sup> Data include demographics, clinical effects, investigations, complications and treatments. The data are entered into the database each week by either trained research staff or medical staff. Each week all admissions are reviewed by a clinical toxicologist and any additional information is obtained directly from the medical record. All patients admitted to the service are seen by the toxicology team daily and treatment is determined by the attending clinical toxicologist. The use of both databases and patient medical records for research has been granted by the respective local area human research ethics committee.

### What is already known

- Pregabalin use is rising worldwide and its abuse potential is being increasingly recognised.
- Despite this there is limited research studying the effects of pregabalin in overdose.

### What this study adds

- Recreational use of pregabalin is increasing.
- Pregabalin overdose in isolation is largely benign, although seizures uncommonly occur.
- Coma is common when sedating agents are co-ingested.

### 2.2 | Patient selection

The databases for both units were searched for all pregabalin exposures presenting from 1 July 2014 to 30 June 2019. Exposure to pregabalin was determined by patient history as part of the toxicological risk assessment.

### 2.3 | Data collection

We extracted data for all pregabalin exposures from the toxicology database. This included baseline characteristics (age, sex), ingestion details (time, dose, intent, co-ingestions), complications (coma [Glasgow Coma Score (GCS) < 9], hypotension [systolic blood pressure (SBP) < 90 mmHg] and seizure), treatment (intubation, vasopressors), disposition, length of stay (LOS) and Intensive Care Unit (ICU) admission.

Medical records were also reviewed for patients taking isolated pregabalin ingestions and recreational ingestions. Data extracted from the medical records included: baseline characteristics (prescribed medications, documented substance use disorder, documented mental health disorder [either Axis I or Axis II condition]), clinical effects (lowest GCS, respiratory rate [RR], lowest SBP) and complications (respiratory depression [RR < 10], seizure duration).

To investigate the relationship between the dose of co-ingested sedative drugs and the occurrence of coma, the diazepam equivalent dose was calculated for all benzodiazepine co-ingestions. Diazepam equivalents were calculated using the multiplication factors of: alprazolam × 5, clonazepam × 10, lorazepam × 5, nitrazepam × 1, oxazepam × 0.17, temazepam × 0.5, zolpidem × 0.5 and zopiclone × 0.67. For opioid co-ingestions, oxycodone was the most common ingested and a comparison was only made for patients co-ingesting oxycodone.

## 2.4 | Analysis

Continuous variables are reported as medians, interquartile ranges (IQR) and ranges. The difference between groups is compared with the Mann–Whitney U test, with a *P*-value of <0.05 considered to be significant. All analysis was performed in GraphPad Prism 8 for Mac OS (GraphPad Software, San Diego, CA, USA; [www.graphpad.com](http://www.graphpad.com)).

## 2.5 | Nomenclature of targets and ligands

Key protein targets and ligands in this article are hyperlinked to corresponding entries in <http://www.guidetopharmacology.org>, the common portal for data from the IUPHAR/BPS Guide to PHARMACOLOGY,<sup>17</sup> and are permanently archived in the Concise Guide to PHARMACOLOGY 2017/18.<sup>18</sup>

## 3 | RESULTS

There were 488 pregabalin poisoning admissions (237 [57%] male) in 413 patients over the five-year period, with 53 patients re-presenting,

some on multiple occasions (median of 1 re-presentation, range: 1–3 re-presentations) (Table 1). The median age was 41 years (IQR 31–50 years, range: 15–89 years). Deliberate self-poisonings accounted for 342 (70%) presentations. There were 121 (25%) recreational exposures in which pregabalin was taken to induce its psychoactive effects. There were 21 (4%) unintentional ingestions (including supratherapeutic ingestions and accidental double dosing), two (0.4%) iatrogenic poisonings and two (0.4%) adverse reactions. The number of presentations increased over the period and excepting an increase overall in the first year, this was then largely due to the increasing number of recreational ingestions from 4% (2/49) to 39% (54/140) (Figure 1).

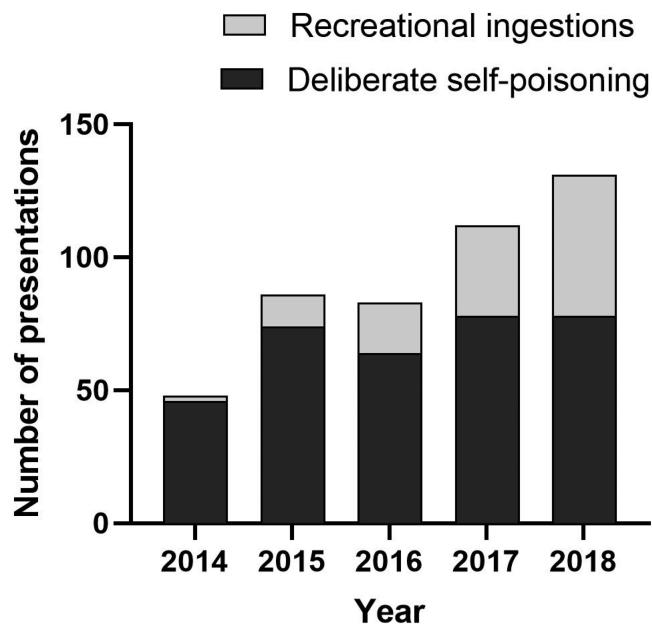
The dose of pregabalin ingested was documented in 396 (81%) cases. The median dose was 1200 mg (IQR 600–3000 mg, range: 75–16 800 mg). Co-ingestions occurred in 427 (88%) presentations, with sedating agents being co-ingested in 387 (79%) presentations; most commonly opioids and benzodiazepines in 201 (41%) and 174 (36%) presentations respectively (Table 2).

Coma occurred in 89 (18%) presentations with a median pregabalin dose of 2400 mg (IQR 875–5250 mg), which was significantly greater among patients without coma (Median 1200 mg; IQR 600–2400 mg; *P* = 0.002). Sedating agents were co-ingested

**TABLE 1** Characteristics of pregabalin poisonings

	All pregabalin exposures <i>n</i> = 488	Pregabalin-only <i>n</i> = 59	Deliberate self-poisonings <i>n</i> = 341	Recreational ingestions <i>n</i> = 121
Number of patients	413	58	299	108
Male	237 (57%)	41 (71%)	151 (52%)	88 (81%)
Median age	41 years	39 years	42 years	36 years
IQR	31–50 years	31–50 years	32–50 years	28–43 years
Range	15–89 years	20–76 years	16–73 years	15–58 years
Pregabalin dose documented	396 (81%)	57 (97%)	296 (87%)	83 (69%)
Median pregabalin dose	1200 mg	1500 mg	1500 mg	900 mg
IQR	600–3000 mg	750–2700 mg	700–3150 mg	600–1500 mg
Range	75–16 800 mg	125–16 800 mg	75–16 800 mg	125–16 800 mg
<b>Clinical findings</b>				
Coma	77 (16%)	1 (2%)	56 (16%)	19 (16%)
Hypotension	26 (5%)	1 (2%)	19 (6%)	4 (3%)
Seizure	10 (2%)	3 (5%)	5 (1%)	4 (3%)
<b>Management</b>				
Intubation	50 (10%)	0	42 (12%)	9 (7%)
Vasopressors	15 (3.1%)	0	15 (4%)	0
<b>Disposition</b>				
Remained in ED	69 (14%)	21 (36%)	53 (16%)	14 (12%)
SSW	339 (70%)	38 (64%)	225 (66%)	97 (80%)
ICU	64 (13%)	0	51 (15%)	9 (7%)
Ward	16 (3%)	0	12 (3%)	1 (1%)
Median length of stay	16.6 h	13.1 h	18.2 h	13.4 h
IQR	10–25 h	6–20 h	12–30 h	9–18 h

ED = emergency department, h = hours, ICU = intensive care unit, IQR = interquartile range, SSW = short stay ward.



**FIGURE 1** Number of pregabalin presentations over five-year period comparing deliberate self-poisoning and recreational ingestions. Each column represents the 12-month period from 1 July in the designated year to 30 June in the subsequent year, corresponding to the Australian financial year

in 83 of these presentations, and in only 6 (1%) patients coma occurred without sedating co-ingestants. In patients with coma taking sedative co-ingestants, the dose of the co-ingested sedating agent was higher in the coma group. In patients that co-ingested benzodiazepines where a dose was available ( $n = 135$ ), those with coma took a median of 163 mg diazepam equivalents (IQR 71–258 mg) vs those without coma that took a median of 50 mg diazepam equivalents (IQR 23–125 mg) ( $P = 0.004$ ). Similarly, in patients that co-ingested oxycodone where a dose was available ( $n = 53$ ), those with coma took a median dose of oxycodone of 135 mg (IQR 88–220 mg) vs 60 mg (IQR 30–115 mg) of oxycodone in those without coma ( $P = 0.11$ ).

Intubation was performed in 52 (11%) presentations. In all cases, sedating co-ingestants were taken. Hypotension occurred in 26 (5%) presentations and in one presentation there was no co-ingestion. Seizures occurred in 11 (2%) cases. The median LOS was 16.5 hours (IQR 10–25 hours). There were 63 (13%) ICU admissions. All patients were discharged home, with 81 (17%) patients having a psychiatric admission prior to discharge.

### 3.1 | Isolated pregabalin ingestion

There were 59 presentations in 58 patients in which pregabalin was taken alone. There were 39 (66%) cases of deliberate self-poisoning, 14 (24%) cases of recreational use, five unintentional exposures and one iatrogenic poisoning. The median dose of pregabalin ingested was 1500 mg (IQR 750–2700 mg).

**TABLE 2** Co-ingestions in pregabalin poisoned patients

	All pregabalin exposures <i>n</i> = 488	Deliberate self-poisonings <i>n</i> = 341	Recreational ingestions <i>n</i> = 121
Any co-ingestion	427 (88%)	299 (88%)	101 (84%)
<b>Sedating co-ingestion</b>	387 (80%)	276 (81%)	88 (73%)
Opioids	200 (41%)	141 (41%)	46 (38%)
Oxycodone	77 (16%)	62 (18%)	6 (5%)
Codeine	41 (9%)	40 (12%)	1 (1%)
Methadone	32 (7%)	17 (5%)	12 (10%)
Heroin	18 (4%)	4 (1%)	14 (12%)
Tramadol	22 (5%)	20 (6%)	2 (2%)
Buprenorphine	16 (3%)	9 (3%)	7 (6%)
Benzodiazepines	176 (36%)	124 (36%)	46 (38%)
Diazepam	107 (22%)	74 (22%)	29 (24%)
Oxazepam	25 (5%)	19 (6%)	5 (4%)
Temazepam	25 (5%)	24 (7%)	0
Alprazolam	20 (4%)	10 (3%)	10 (8%)
Clonazepam	15 (3%)	10 (3%)	5 (4%)
Alcohol	86 (18%)	64 (19%)	20 (17%)
Quetiapine	47 (10%)	37 (11%)	6 (5%)
TCA	29 (6%)	24 (7%)	4 (3%)
Mirtazapine	25 (5%)	24 (7%)	1 (1%)
Olanzapine	15 (3%)	13 (4%)	1 (1%)
Baclofen	14 (3%)	7 (2%)	6 (5%)
<b>Non-sedating co-ingestion</b>	216 (44%)	167 (49%)	38 (31%)
Paracetamol	77 (16%)	72 (21%)	2 (2%)
SSRI/SNRI	75 (15%)	67 (20%)	1 (1%)
Methamphetamine	41 (8%)	7 (2%)	33 (27%)
NSAID	34 (7%)	29 (9%)	0

NSAID = Non-steroidal anti-inflammatory drug, SNRI = Serotonin noradrenaline reuptake inhibitor, SSRI = Selective serotonin reuptake inhibitor, TCA = Tricyclic antidepressants.

The median lowest GCS was 14 (IQR 13–15, range 6–15). Only one patient developed coma and had a GCS of 6 (motor score [M] 4), 7 hours after ingesting 2400 mg of pregabalin to which he was naïve. He was managed supportively, and his GCS improved to 14 over 4 hours. No patient developed respiratory depression. Hypotension occurred in one patient following the administration of droperidol for agitation, which was managed with intravenous fluids. There were three patients who had seizures, following ingestions of 600 mg, 900 mg and 1800 mg of pregabalin. One patient ingesting 600 mg had a pre-existing seizure disorder. All seizures were self-limiting and lasted approximately 1 minute.

### 3.2 | Recreational ingestions

There were 121 presentations in 108 patients in which pregabalin was used recreationally. Pregabalin was prescribed in 51/108 (47%) patients, with a median daily dose of 600 mg (range: 75–1200 mg). Opioids and benzodiazepines were prescribed medications in 45 (42%) and 48 (44%) patients respectively. An existing substance use disorder was documented in 107/108 patients, and 50 of these were prescribed pregabalin. Sixty-one of the 108 (56%) patients had a documented history of mental illness. The median dose of pregabalin ingested was 900 mg (IQR 600–1500 mg, range 125–16 800 mg), which was less than the dose taken in deliberate self-poisoning. Sedating agents were co-ingested in 89 (74%) presentations (Table 2).

## 4 | DISCUSSION

Pregabalin poisoning is increasing, much of which can be attributed to a rapid rise in recreational use. Overall, pregabalin overdose did not cause severe toxicity, with mild sedation and uncommonly seizures. Co-ingestion of medications was associated with more severe toxicity, in particular co-ingestion of opioids and benzodiazepines were associated with coma.

Our findings are similar to previous reports which found isolated ingestions to be benign,<sup>12,13</sup> although we identified that self-limiting seizures are an uncommon but potentially important effect. It appears that, similar to other anticonvulsants such as lamotrigine and carbamazepine, pregabalin has proconvulsant properties in overdose.

Pregabalin is usually not taken in isolation but rather as part of a sedating polypharmacy ingestion. In combination with other sedating agents, pregabalin appears to have an additive depressant effect. Similar to our results, a retrospective study of 1208 ambulance attendances with pregabalin misuse found 68% co-ingested a sedating agent with the commonest being benzodiazepines and opioids.<sup>9</sup> The combination of pregabalin and opioids or benzodiazepines is being implicated in an increasing number of deaths.<sup>12</sup>

Interestingly, we did find that coma was associated with larger doses of pregabalin, which on the face of it would suggest that pregabalin causes dose-dependent central nervous system depression. However, coma was rare in pregabalin-only ingestions. In patients taking sedating co-ingestions, coma was associated with larger doses of co-ingested benzodiazepines and oxycodone. This suggests that severe effects occurred in patients who took larger ingestions overall, with the increased pregabalin dose being a surrogate marker of a large total tablet ingestion.

The rising number of pregabalin exposures correlates with the rising prescription rate.<sup>4,9</sup> Since becoming subsidised on the Australian Pharmaceutical Benefit Scheme in March 2013, pregabalin prescriptions numbered approximately 3.75 million in 2017–2018 making it the sixth top subsidised drug on the scheme.<sup>17</sup>

The rising prescription rate is occurring in developed countries across the world. In England over the same period, pregabalin

prescriptions have more than doubled, from 3.3 million in 2013 to 7.0 million in 2018.<sup>18</sup> Our study found presentations per annum over the five-year period increased almost three-fold, and much of this rise was due to increased recreational use.

The potential for pregabalin misuse has been recognised for some time. Some health authorities, like Public Health England in conjunction with the National Health Service released advice to prescribers warning of the potential of gapapentinoid misuse over 5 years ago.<sup>19</sup> It is unclear if this message is reaching prescribers given the rising prescription rate and increasing implication of pregabalin in poisoning deaths.<sup>2,4,11</sup> Concerningly, in this study we found that pregabalin is often prescribed to vulnerable populations, which includes people with substance use disorders and documented mental health illness. Similar to our findings, Cairns et al. reported that one in seven Australian patients dispensed pregabalin appear at high risk of misuse, with 66% of poisoned patients having a documented substance use history.<sup>4</sup>

There are a number of limitations of this study, the main one being its retrospective design and the potential inaccuracies in the documented risk assessment. However, both toxicology units prospectively enter all admissions into a clinical database, which includes detailed information on the most important clinical effects, complications and treatments. There is a possibility that novel or unusual effects will not be reported, but we additionally reviewed the medical records of pregabalin-only presentations.

Another potential limitation is that pregabalin ingestion was not confirmed by analytical testing, with the dose of pregabalin and any co-ingestion relying on the patient report. The accuracy of patient history has been reported previously, including in pharmacokinetic studies of overdose.<sup>20,21</sup>

A final limitation was the ability of the study to determine the underlying reason for increased sedative effects: whether this was a synergistic effect of pregabalin and other sedative medications, or simply the fact that these patients ingested a number of sedative medications, and an increased dose of pregabalin was simply associated with an increased dose of other sedatives, such as benzodiazepines and oxycodone.

## 5 | CONCLUSION

Deliberate self-poisoning with pregabalin in isolation is largely benign, resulting in mild sedation and uncommonly seizures. Pregabalin is more commonly taken in combination with other sedating agents, increasing the likelihood of coma and respiratory depression. Recreational exposure is increasingly common, and its abuse and misuse potential should not be underestimated. Caution needs to be exercised when prescribing pregabalin to patients with a history of substance misuse.

## ACKNOWLEDGEMENT

G.K. Isbister is funded by an NHMRC Senior Research Fellowship ID1061041.

## COMPETING INTERESTS

There are no competing interests to declare.

## CONTRIBUTORS

K.Z.I. and G.K.I. conceived the study and analysed the data. K.Z.I., G.P. and K.H. completed data collection. K.Z.I. and G.K.I. analysed the data and performed statistical analysis. K.Z.I. drafted the manuscript, and all authors contributed substantially to its revision. K.Z.I. takes responsibility for the paper as a whole.

## DATA AVAILABILITY STATEMENT

Data available on request from the authors.

## ORCID

Katherine Z. Isoardi  <https://orcid.org/0000-0002-1176-7923>

Geoffrey K. Isbister  <https://orcid.org/0000-0003-1519-7419>

## REFERENCES

- Bonnet U, Scherbaum N. How addictive are gabapentin and pregabalin? A systematic review. *Eur Neuropsychopharmacol*. 2017;27: 1185-1215. <https://doi.org/10.1016/j.euroneuro.2017.08.430>
- Evoy K, Morrison M, Saklad S. Abuse and misuse of pregabalin and gabapentin. *Drugs*. 2017;77(4):403-426. <https://doi.org/10.1007/s40265-017-0700-x>
- Lyndon A, Audrey S, Wells C, et al. Risk to heroin users of poly-drug use of pregabalin or gabapentin. *Addiction*. 2017;112(9):1580-1589. <https://doi.org/10.1111/add.13843>
- Cairns R, Schaffer A, Ryan N, Pearson S, Buckley N. Rising pregabalin use and misuse in Australia: trends in utilisation and intentional poisonings. *Addiction*. 2018;114(6):1026-1034. <https://doi.org/10.1111/add.14412>
- Schjerning O, Pottegård A, Damkier P, Rosenzweig M, Nielsen J. Use of pregabalin – a nationwide pharmacoepidemiological drug utilization study with focus on abuse potential. *Pharmacopsychiatry*. 2016;49: 155-161. <https://doi.org/10.1055/s-0042-101868>
- Johansen M. Gabapentinoid use in the United States 2002 through 2005. *JAMA Intern Med*. 2018;178(2):292-294. <https://doi.org/10.1001/jamainternmed.2017.7856>
- Buckley NA, Whyte IM, Dawson AH, Isbister GK. A prospective cohort study of trends in poisoning, Newcastle, Australia, 1987-2012: plus ça change, plus c'est la même chose. *Med J Aust*. 2015;202(8): 438-443. <https://doi.org/10.5694/mja14.01116>
- Daly C, Griffin E, Ashcroft D, Webb R, Perry I, Arensman E. Intentional drug overdose involving pregabalin and gabapentin: findings from the National Self-Harm Registry Ireland, 2007-2015. *Clin Drug Investig*. 2018;38(4):373-380. <https://doi.org/10.1007/s40261-017-0616-y>
- Crossin R, Scott D, Arunogiri S, Smith K, Dietze P, Lubman DL. Pregabalin misuse-related ambulance attendances in Victoria, 2012-2017: characteristics of patients and attendances. *Med J Aust*. 2019;210(2):75-79. <https://doi.org/10.5694/mja2.12036>
- Lyrica® (pregabalin) Product Information leaflet. Sydney: Pfizer Australia. Version pfplyric11019. Revised 11 October 2019. Available at: <http://secure.healthlinks.net.au/content/pf/retriever.cfm?product=pfplyric10113>. Accessed 22 October 2019.
- Abrahamsson T, Berge J, Ojehagen A, Hakansson A. Benzodiazepine, z-drug and pregabalin prescriptions and mortality among patients in opioid maintenance treatment – a nationwide register-based open cohort study. *Drugs Alcohol Dependence*. 2017;174:58-64. <https://doi.org/10.1016/j.drugalcdep.2017.01.013>
- Spiller H, Bratcher R, Griffith J. Pregabalin overdose with benign outcome. *Clin Toxicol*. 2008;46(9):917. <https://doi.org/10.1080/15563650801986497>
- Wills B, Reynolds P, Chu E, et al. Clinical outcomes in newer anticonvulsant overdose: a poisons center observational study. *J Med Toxicol*. 2014;10(3):254-260. <https://doi.org/10.1007/s13181-014-0384-5>
- Woods D, Berry D, Glover G, Eastwood J, Dargan P. Significant pregabalin toxicity managed with supportive care alone. *J Med Toxicol*. 2010;6(4):435-437. <https://doi.org/10.1007/s13181-010-0052-3>
- Braga A, Chidley K. Self-poisoning with lamotrigine and pregabalin. *Anaesthesia*. 2007;62(5):524-527. <https://doi.org/10.1111/j.1365-2044.2006.04913.x>
- Downes MA, Page CB, Berling I, Whyte IM, Isbister GK. Use of a tablet-based application for clinical handover and data collection. *Clin Toxicol*. 2019;11:1-6. <https://doi.org/10.1080/15563650.2019.1674322> [EPub ahead of print]
- Pharmaceutical Benefits Scheme (PBS). Expenditure and Prescriptions Twelve Months to 30 June 2018. Canberra: PBS; 201. Available at: <https://www.pbs.gov.au/statistics/expenditure-prescriptions/2017-2018/expenditure-and-prescriptions-twelve-months-to-30-june-2018.pdf>. Accessed 22 October 2019.
- Prescription Cost Analysis. – England 2013 and England 2018. NHS England. <https://digital.nhs.uk/data-and-information/publications/statistical/prescription-cost-analysis>. Accessed 16 March 2020.
- Advice for prescribers on the risk of the misuse of pregabalin and gabapentin. December 2014. Public Health England and NHS England. [https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment\\_data/file/385791/PHE-NHS\\_England\\_pregabalin\\_and\\_gabapentin\\_advice\\_Dec\\_2014.pdf](https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/385791/PHE-NHS_England_pregabalin_and_gabapentin_advice_Dec_2014.pdf). Accessed 16 March 2020.
- Friberg LE, Isbister GK, Duffull SB. Pharmacokinetic-pharmacodynamic modelling of QT interval prolongation following citalopram overdose. *Br J Clin Pharmacol*. 2006;61(2):177-190. <https://doi.org/10.1111/j.1365-2125.2005.02546.x>
- Isbister GK, Friberg LE, Stokes B, et al. Activated charcoal decreases the risk of QT prolongation after citalopram overdose. *Ann Emerg Med*. 2007;50(5):593-600. <https://doi.org/10.1016/j.annemergmed.2007.03.009>

**How to cite this article:** Isoardi KZ, Polkinghorne G, Harris K, Isbister GK. Pregabalin poisoning and rising recreational use: a retrospective observational series. *Br J Clin Pharmacol*. 2020; 86:2435-2440. <https://doi.org/10.1111/bcp.14348>